Intensive Care Med (2018) 44:2235–2238
https://doi.org/10.1007/s00134-018-5145-5

UNDERSTANDING THE DISEASE

Understanding spontaneous vs. ventilator breaths: impact and monitoring

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Introduction

Spontaneous breathing during mechanical ventilation balances important advantages including improved oxygenation [1] and less diaphragm disuse [2] against serious disadvantages including increased injury to the lung and diaphragm [2–5] and potentially lower survival [6]. Of course, spontaneous breathing is an absolute requirement for successful weaning, and so it must ultimately be a goal in all patients. While the traditional focus in acute respiratory distress syndrome (ARDS) is on controlling and monitoring ventilator breaths, recent advances point to important differences between spontaneous and mechanical breaths in terms of pathophysiology and monitoring [3, 4, 7]. This paper reviews these insights and provides suggestions for bedside monitoring of spontaneous effort in patients with ARDS during mechanical ventilation, focusing on the use of esophageal manometry.

Monitoring mechanical breaths

During a mechanical breath (i.e., without spontaneous effort), ventilation is preferentially distributed to the non-dependent "baby" lung, in part because of the predominance of atelectasis in the dependent lung; this explains why, during paralysis, the non-dependent "baby" lung is one of the regions more susceptible to stretch-induced injury [8–10]. In order to avoid such injury, physicians attempt to limit tidal volume (V_T) or airway pressure (P_{aw}), or in some cases, the transpulmonary pressure (P_L) [7, 11].

P_{aw} consists of two components: resistive pressure, which generates airflow through the airways and/or relates to tissue resistance and the endotracheal tube; and, alveolar pressure, which distends the alveoli and chest wall [11, 12]. Peak P_{aw} comprises resistive and alveolar components. At end-inspiration airflow has ceased, and because there is now no "resistive" component, the resulting "plateau" pressure reflects the pressure distending the alveoli and chest wall [11, 12]. Therefore, plateau phase, either P_{aw}, or P_L, e.g., plateau P_{aw}, driving pressure, or plateau P_L, and not peak phase, best reflects the maximal stretch of distended alveoli (Fig. 1a)—and their propensity to injury, and for this reason clinicians target plateau P_{aw} to less than 30 cmH_2O (or plateau P_L to less than 25 cmH_2O) to prevent ventilator-induced lung injury [7, 11]. The relationships among pressures (peak and plateau, airway and transpulmonary) and regional lung distension during a mechanical breath are illustrated in Fig. 1a.

Monitoring spontaneous breaths

The context is more complicated during spontaneous effort for several reasons. First, the addition of spontaneous effort to a mechanical breath involves a (negative) deflection in pleural pressure (P_{pl}) combined with a (positive) deflection in P_{aw}, which results in an additive increase in the distending pressure (i.e., $P_L = P_{aw} - P_{pl}$). Therefore, reliance on P_{aw} (plateau P_{aw} or driving pressure) is not sufficient to limit injurious stretch; instead, esophageal pressure (P_{es}) can be measured to assess the intensity of the effort, i.e., the negative deflection (or "swing") in P_{es} caused by the effort, and to calculate the P_L [7, 13]. Second, spontaneous effort exerts its impact differently on the non-dependent vs. the dependent lung. The plateau phase of P_L is associated with maximal stretch in the non-dependent lung, but not the dependent lung. When peak P_L occurs at the time that P_{es} is most negative as a result of vigorous effort, peak P_L could correspond to time of maximal distension in the dependent

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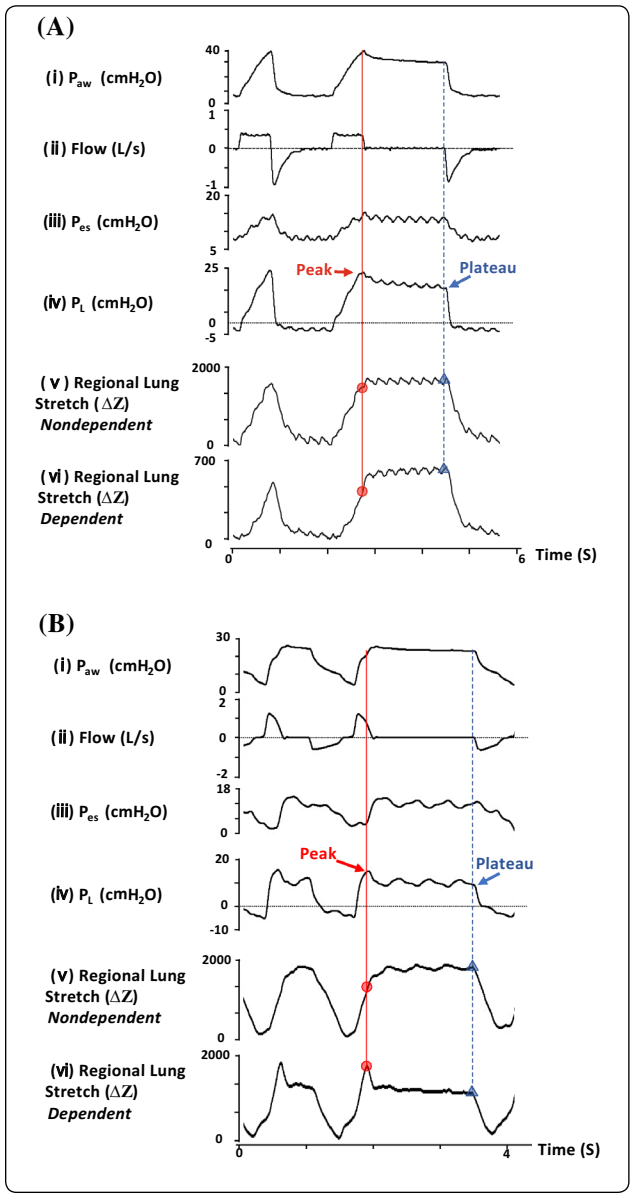

Fig. 1 **a** Local inflation pattern during a mechanical breath: this is a representative description in a severe ARDS porcine model (i.e., repeated surfactant depletion + injurious mechanical ventilation). Two consecutive breaths are presented: the first, without interruption, and the second with an inspiratory hold. Volume-controlled ventilation with square flow was provided, as indicated in the tracings of P_{aw} (i) and flow (ii). There were no negative deflections in P_{es} (iii). P_L was calculated as [$P_{aw} - P_{es}$] (iv), and regional lung stretch (ΔZ) was determined using electrical impedance tomography (EIT; PulmoVista®500, Dräger, Lübeck, Germany) with the thorax divided into two zones: non-dependent and dependent (v and vi, respectively). Maximum inflation of the non-dependent and dependent lung (v and vi) was achieved at end-inspiration (i.e., plateau phase, blue line), but not at peak phase (red line). **b** Local inflation pattern during a spontaneous effort: this is a representative description in a severe ARDS porcine model (i.e., repeated surfactant depletion + injurious mechanical ventilation). Two consecutive breaths are presented: the first, without interruption, and the second with an inspiratory hold. The presence of spontaneous breathing caused a negative deflection in P_{aw} (i) and increased tracheal gas flow after triggering (ii), resulting from a negative deflection in P_{es} (iii). Early in inspiration, peak P_L (iv) occurred, corresponding to the time when the swing in P_{es} from spontaneous effort reached most negative, but not corresponding to the time when P_{aw} reached peak. This is contrast to the findings during muscle paralysis (**a**). Non-dependent (v) and dependent lung (vi) show local stretch, reflected by delta Z (i.e., relative change in air content). Peak P_L corresponds to the duration of maximum inflation of the dependent lung (red dot in vi), but not the non-dependent lung (red dot in v). Moreover, the maximal stretch in the dependent lung occurred despite the presence of residual inspiratory tracheal gas flow (ii). When the P_{es} began to rise, corresponding to the start of diaphragm relaxation, the dependent lung began to deflate (vi) and the gas moved into non-dependent lung (v), together with residual inspiratory flow from the ventilator. As a result, the non-dependent lung continued to inflate (v) and its stretch was maximal at end-inspiration (blue triangle in v). P_{aw} airway pressure, P_{es} esophageal pressure, P_L transpulmonary pressure

lung (Fig. 1b). Therefore, in contrast to mechanical breaths, plateau P_L (i.e., at end-inspiration) potentially underestimates maximal dependent lung stress/stretch during vigorous effort in ventilated patients with ARDS. Third, vigorous effort appears to increase injury in the dependent lung—the same region in which spontaneous effort increases inspiratory distension [4].

The key mechanism is inhomogeneous pressure transmission in the presence of "solid-like" injured lung (Supplemental Figure). Here, the negative deflection in P_{pl} resulting from diaphragm contraction is poorly transmitted to the remainder of the pleural surface, and thus "confined" to the dependent lung [3, 4, 14]. The higher distending pressure in the local lung will tend to draw gas from the non-dependent lung (this is called pendelluft [14]), or from the trachea and ventilator, towards the dependent lung. This causes a transient overdistension [3, 4, 14] and tidal recruitment in the dependent lung [3, 4] during early inspiration (i.e., the peak phase of P_L), corresponding, in space and time, to maximal intensity of the diaphragm contraction and the peak negative value of deflection (swing) in P_{es}. Importantly, such injurious inflation is likely observed in the presence of vigorous effort, and "solid-like" atelectatic lung tissue due to insufficient PEEP [4, 5, 15].

Clinical implications

Limitations of V_T and P_{aw} are validated clinical approaches to lessen ventilator-induced lung injury during ventilator breaths, but effort-dependent lung injury is not preventable using such global parameters [3, 4, 14].

Instead monitoring P_L and P_{es} may be preferable during spontaneous breaths, especially when spontaneous effort is vigorous. First, here plateau P_L (at end-inspiration) corresponds to time of maximal distension in the non-dependent lung, but this is not always a good surrogate for dependent lung stress. When peak P_L occurs at the time that P_{es} is most negative as a result of vigorous effort, (even with residual inspiratory flow) it is important to note that peak P_L could correspond to time of maximal distension in the dependent lung—the region most at risk during spontaneous effort. Thus, the limitation of peak P_L as well as plateau P_L may become an important target in preventing effort-dependent lung injury. In future, we might carefully evaluate the safe upper limit of P_L (or ΔP_L) calculated using esophageal balloon manometry in order to minimize effort-dependent lung injury. This is because the assessment using P_{es} (i.e., ΔP_L or P_L) could misrepresent the "true" P_L in the dependent lung due to "solid-like" dependent lung behavior and a vertical gradient of P_{pl}. Second, monitoring the degree of negative "swing" in P_{es} (i.e., intensity of spontaneous effort) can facilitate a balance between avoiding diaphragm disuse (from absence of effort) and overuse injury (from excess effort), thereby preventing ventilator-induced diaphragm dysfunction [2]. Third, P_{es} is useful to monitor patient–ventilator asynchronies and to estimate vascular distending pressures, which are potentially related to effort-dependent lung injury [7]. Finally, although regional pressure measurements are of increasing interest, they might ultimately best be assessed in conjunction with real-time regional lung imaging.

Conclusion

Emerging insights into the pathophysiology of spontaneous effort during mechanical ventilation are not intuitive but can be better understood with bedside monitoring such as esophageal manometry. Subsequent studies will determine the validity of—and identify thresholds for—titration of peak and plateau P_L, as well as swings in P_{es}, in best protecting the lungs and diaphragms of patients with ARDS.

Electronic supplementary material

The online version of this article (https://doi.org/10.1007/s00134-018-5145-5) contains supplementary material, which is available to authorized users.

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Compliance with ethical standards

Conflicts of interest

TY and BPK have applied for a patent on a CNAP (continuous negative abdominal pressure) device.

Received: 12 February 2018 Accepted: 19 March 2018
Published online: 24 March 2018

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